14.1 The Physiology of Stress and Stress-Related Illness

AQA 7182 · about 50 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Outline a behavioural mediator such as increased smoking, poor diet or reduced exercise linking chronic stress to heart disease.

(Total for Question 1 is 2 marks)

2 Outline the sympathomedullary pathway, naming medulla, sympathetic nerves, adrenal medulla and the catecholamines.

(Total for Question 2 is 3 marks)

3 Outline evidence for a link between chronic stress and cardiovascular disease, citing a physiological marker such as hypertension or raised cholesterol.

(Total for Question 3 is 2 marks)

4 Outline two applications such as workplace stress reduction policies, early screening for cardiovascular risk, or interventions to improve sleep and immune function.

(Total for Question 4 is 2 marks)

5 Outline the three stages of Selye's GAS: alarm, resistance and exhaustion, with a brief feature of each.

(Total for Question 5 is 3 marks)

6 Explain a methodological and conceptual limitation of GAS such as animal-based evidence and ignoring cognitive appraisal.

(Total for Question 6 is 2 marks)

7 Outline a methodological strength such as experimental wound procedure or objective physiological measures in Kiecolt-Glaser research.

(Total for Question 7 is 2 marks)

8 Discuss the physiology of stress and its role in illness, using theory and empirical evidence and evaluating strengths and limitations.

(Total for Question 8 is 16 marks)

9 Apply stress physiology and Kiecolt-Glaser findings to explain Priya's slow wound healing and give one limitation of causal inference.

(Total for Question 9 is 6 marks)

Mark scheme · 14.1 The Physiology of Stress and Stress-Related Illness

Question 1

identifies a behavioural change such as increased smoking, overeating or reduced exercise under chronic stress oe **B1**

explains that such behaviours raise cardiovascular risk by increasing cholesterol, blood pressure or obesity oe **B1**

Answer: Chronic stress can lead to greater smoking, poor diet and less exercise, which raise cholesterol and blood pressure and increase obesity, thereby elevating cardiovascular risk.

Question 2

identifies the pathway as involving the sympathetic nervous system and the adrenal medulla oe **M1**

states the adrenal medulla releases adrenaline and noradrenaline (epinephrine and norepinephrine) oe **A1**

explains this produces the fight-or-flight changes such as increased heart rate and blood flow to muscles oe **A1**

Answer: Sympathetic nervous system activation stimulates the adrenal medulla to release adrenaline and noradrenaline, producing rapid fight-or-flight changes such as increased heart rate and mobilisation of energy.

Question 3

identifies evidence linking chronic stress with cardiovascular risk, e.g. studies showing chronic stress is associated with raised blood pressure or hypertension oe **B1**

states that raised blood pressure or raised cortisol combined with behavioural factors increases risk of heart disease oe **B1**

Answer: Studies show chronic stress is associated with raised blood pressure, and sustained hypertension is a risk marker for cardiovascular disease; chronic cortisol and behaviour changes can raise this risk.

Question 4

outlines one application, e.g. workplace stress reduction programmes to lower chronic cortisol exposure and reduce illness risk oe **M1**

outlines a second application, e.g. screening and monitoring of blood pressure and lifestyle interventions for stressed patients to prevent cardiovascular disease oe **M1**

Answer: Applications include workplace stress reduction policies to reduce chronic cortisol exposure and public health screening of stressed individuals for high blood pressure and lifestyle interventions to prevent cardiovascular disease.

Question 5

names the three stages: alarm, resistance and exhaustion oe **M1**

explains alarm is the immediate fight-or-flight response involving adrenaline/cortisol oe **A1**

explains resistance is where physiological responses are maintained to cope, and exhaustion follows when resources are depleted leading to illness oe **A1**

Answer: GAS stages: alarm (immediate fight-or-flight response), resistance (sustained physiological arousal to cope) and exhaustion (resources depleted leading to vulnerability to illness).

Question 6

identifies reliance on animal studies or that GAS treats stress responses as uniform rather than influenced by appraisal oe **M1**

explains why this matters, e.g. animal physiology and stressors may not generalise to human psychological stress and cognitive appraisal can alter hormonal responses oe **A1**

Answer: GAS is based largely on animal experiments so findings may not generalise to humans whose stress responses are influenced by cognition and appraisal; this matters because human psychological factors can change hormonal responses and the course of stress-related illness.

Question 7

identifies a strength: use of a standardised, experimental wound procedure and objective physiological measures of healing oe **M1**

explains that such objective measures increase internal validity by directly measuring biological outcomes rather than relying on self-report oe **A1**

Answer: A strength was using a standardised wound and objective measures of healing, which increases internal validity by providing direct biological evidence that stress can impair immune function.

Question 8

Level 4 Knowledge of stress physiology and stress-related illness is detailed and accurate. Discussion is thorough and evaluative, integrating theory and empirical evidence, and considers methodological issues, alternative explanations and real-world implications. Specific studies and mechanisms are applied to support points and evaluation is well developed and coherent, with clear specialist terminology. **13-16**

Level 3 Knowledge is mostly accurate and relevant. Discussion includes strengths and limitations and refers to evidence and theory, but evaluation may be less thorough or slightly imbalanced. Some application to studies or implications is present. Specialist terms are used appropriately. **9-12**

Level 2 Knowledge is present but limited in depth or detail. Discussion contains some evaluative points but these are simple or not well developed. Limited use of evidence or theory to support claims. Answer may be unstructured in places. **5-8**

Level 1 Knowledge is minimal and answers are brief or fragmented. Little or no evaluation. Little use of relevant studies or theory. Specialist terminology may be missing or misused. **1-4**

Level 0 No creditable content. **0**

Indicative content:

- AO1: Describe physiological systems involved in stress: the sympathomedullary pathway producing adrenaline and noradrenaline for immediate fight-or-flight changes, and the HPA axis releasing cortisol for longer term stress responses.
- AO1: Outline Selye's general adaptation syndrome with its alarm, resistance and exhaustion stages as a model linking prolonged stress to illness.
- AO1: Describe mechanisms by which chronic cortisol and sympathetic activation can cause illness: immune suppression, raised blood pressure, metabolic changes and increased atherosclerosis risk.
- AO2: Kiecolt-Glaser et al. wound-healing studies provide empirical evidence that people under chronic stress or caregiving burden heal more slowly, demonstrating immune impact.
- AO3: Evaluate Selye's GAS: initial strength was unifying biological model and experimental animal evidence, but limitations include reliance on animal studies, simplistic stage model and lack of psychological appraisal; human stress responses are moderated by cognition and context.
- AO3: Evaluate Kiecolt-Glaser: strength is standardised wound procedure and objective immune measures, but many stress-illness studies are correlational and confounding variables such as health behaviours or medication may influence results, limiting causal claims.
- AO3: Consider individual differences in cortisol reactivity and coping styles; some individuals show exaggerated cortisol responses, others blunted responses, which affects vulnerability to illness and challenges universal predictions.
- AO3: Consider methodological issues such as ecological validity of laboratory stressors versus chronic real-world stress, ethical constraints on inducing long-term stress, and temporal validity of longitudinal versus cross-sectional designs.
- AO3: Practical implications include interventions to reduce chronic stress for public health, early screening for cardiovascular risk in high stress occupations and targeted support for vulnerable individuals, but applications must consider individual differences and the multifactorial nature of illness.
- AO3: Conclude by weighing evidence: physiological pathways plausibly link stress to illness, supported by experimental and longitudinal studies, yet causal claims require caution because of correlational evidence, individual variation and complexity of biopsychosocial interactions.

Question 9

links chronic stress to elevated cortisol levels which can suppress immune function oe	**M1**
explains that suppressed immune function reduces leukocyte activity and cytokine signalling needed for tissue repair, slowing healing oe	**A1**
refers to Kiecolt-Glaser type evidence showing stressed caregivers had slower wound healing in an experimental wound study oe	**M1**
applies this to Priya by noting her long shifts and poor sleep resemble chronic stressors that could replicate the slower healing effect oe	**A1**
identifies an additional physiological mechanism such as elevated sympathetic activity increasing blood pressure which can impair microcirculation to the wound oe	**M1**
gives a limitation: many stress-illness studies are correlational so other factors like nutrition or medication could confound the association between stress and healing oe	**A1**

Answer: Chronic stress elevates cortisol which suppresses immune cells and cytokines needed for repair, and sympathetic overactivity can impair microcirculation, both slowing healing. Kiecolt-Glaser found caregivers healed more slowly, supporting this. However the research is often correlational and other factors such as sleep, diet, medication or smoking could confound the relationship, so causation is not certain.